

WEST Search History

DATE: Friday, August 04, 2006

<u>Hide?</u>	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
<i>DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L4	L3 and endotheli\$	17
<input type="checkbox"/>	L3	GPR39 or GPR 39	45
<input type="checkbox"/>	L2	L1 and endothelia\$	2
<input type="checkbox"/>	L1	GPCR 4941 or GPCR4941	2

END OF SEARCH HISTORY

\$\$\$STN;HighlightOn= ***;HighlightOff=*** ;

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspla1633cxq

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

***** Welcome to STN International *****

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 "Ask CAS" for self-help around the dock
NEWS 3 FEB 27 New STN AnaVist pricing effective March 1, 2006
NEWS 4 APR 04 STN AnaVist \$500 visualization usage credit offered
NEWS 5 MAY 10 CA/CAPLUS enhanced with 1900-1906 U.S. patent records
NEWS 6 MAY 11 KOREAPAT updates resume
NEWS 7 MAY 19 Derwent World Patents Index to be reloaded and enhanced
NEWS 8 MAY 30 IPC 8 Rolled-up Core codes added to CA/CAPLUS and
USPATFULL/USPAT2
NEWS 9 MAY 30 The F-Term thesaurus is now available in CA/CAPLUS
NEWS 10 JUN 02 The first reclassification of IPC codes now complete in
INPADOC
NEWS 11 JUN 26 TULSA/TULSA2 reloaded and enhanced with new search and
display fields
NEWS 12 JUN 28 Price changes in full-text patent databases EPFULL and
PCTFULL
NEWS 13 JUL 11 CHEMSAFE reloaded and enhanced
NEWS 14 JUL 14 FSTA enhanced with Japanese patents
NEWS 15 JUL 19 Coverage of Research Disclosure reinstated in DWPI

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b,
CURRENT

MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP).
AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS LOGIN Welcome Banner and News Items
NEWS IPC8 For general information regarding STN implementation of IPC 8
NEWS X25 X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that
specific topic.

All use of STN is subject to the provisions of the STN Customer
agreement. Please note that this agreement limits use to scientific
research. Use for software development or design or implementation
of commercial gateways or other similar uses is prohibited and may
result in loss of user privileges and other penalties.

***** STN Columbus *****

FILE 'HOME' ENTERED AT 16:52:42 ON 27 JUL 2006

=>

=>

COST IN U.S. DOLLARS	ENTRY	SESSION	TOTAL
FULL ESTIMATED COST	12.81	12.81	

FILE 'EMBASE' ENTERED AT 17:29:05 ON 27 JUL 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 17:29:05 ON 27 JUL 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 17:29:05 ON 27 JUL 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> s GCPR (3a) 4941
L1 0 GCPR (3A) 4941

=> s GPCR (3a) 4941
L2 1 GPCR (3A) 4941

=> d bib abs

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:798478 CAPLUS <<LOGINID::20060727>>
DN 135:340279
TI A novel G protein-coupled receptor sequence homolog 4941, and related
methods and compositions for the diagnosis and treatment of cardiovascular
and tumorigenic disease
IN Galvin, Katherine A.; Rudolph-Owen, Laura A.

PA Millennium Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 118 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001081634	A2	20011101	WO 2001-US13788	20010425
WO 2001081634	A3	20020912		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG AU 2001057406 A5 20011107 AU 2001-57406 20010425 EP 1280937 A2 20030205 EP 2001-930917 20010425 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR US 2004091929 A1 20040513 US 2003-696706 20031029 PRAI US 2000-199908P P 20000426 US 2000-635521 A 20000809 WO 2001-US13788 W 20010425 AB The invention provides protein and cDNA sequences for a novel human G protein-coupled receptor ("GPCR") sequence homolog "4941" "GPCR" "4941" gene is located at human chromosome 2q21-22 and its mRNA tissue profile is provided. Specifically, the present invention identifies "GPCR" "4941" genes which are differentially expressed in cardiovascular disease states, relative to their expression in normal, or non-cardiovascular disease states, and/or in response to manipulations relevant to cardiovascular disease. The present invention also identifies "GPCR" "4941" genes as differentially expressed in tumorigenic disease, e.g., ovarian cancer. The present invention relates to methods and compns. for the diagnosis and treatment of cardiovascular disease and cancers. These diseases include but not limit, atherosclerosis reperfusion injury, hypertension, restenosis, arterial inflammation, and endothelial cell disorders, such as disorders assoc. with aberrant endothelial cell growth, angiogenesis and/or vascularization, e.g., tumorigenic disorders. The present invention describes methods for the diagnostic evaluation and prognosis of various cardiovascular and tumorigenic diseases, and for the identification of subjects exhibiting a predisposition to such conditions. The present invention provides methods for the diagnostic monitoring of patients undergoing clin. evaluation for the treatment of cardiovascular disease and tumorigenic, and for monitoring the efficacy of compds. in clin. trials. The present invention also provides methods for the identification and therapeutic use of compds. as treatments of cardiovascular and tumorigenic disease.				

=> s GPCR and endothelia?

L3 216 GPCR AND ENDOTHELIA?

=> dup rem l3

PROCESSING COMPLETED FOR L3

L4 132 DUP REM L3 (84 DUPLICATES REMOVED)

=> s l4 and py<=2000

L5 13 L4 AND PY<=2000

=> d bib abs 1-

YOU HAVE REQUESTED DATA FROM 13 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN

AN 2001096418 EMBASE <<LOGINID::20060727>>

TI A subfamily of G protein-coupled cellular receptors for lysophospholipids
and lysosphingolipids.

AU Goetzl E.J.; An S.

CS E.J. Goetzl, Department of Medicine, Univ. of California Medical Center,
San Francisco, CA 94143-0711, United States

SO Advances in Experimental Medicine and Biology, (2000) Vol. 469, pp.
259-264.

Refs: 12

ISSN: 0065-2598 CODEN: AEMBAP

CY United States

DT Journal; Conference Article

FS 029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 29 Mar 2001

Last Updated on STN: 29 Mar 2001

AB The results of molecular cloning and homology searches have identified a
minimum of five different proteins of the "endothelial"
differentiation gene (edg) encoded subfamily of GPCRs. Edg protein GPCRs
show amino acid sequence identity of 31% to 34% as a subfamily, but
contain two homology clusters with greater similarity of structures and
functions. One cluster of high amino acid sequence homology includes
Edg-2 and Edg-4 proteins, that encode GPCRs for LPA, but not

lysophospholipids. A second homology cluster encompasses Edg-1, Edg-3 and Edg-5. Edg-3 and Edg-5 encode GPCRs specific for S1P, but not LPA. Preliminary data suggest that Edg-1 encodes a ***GPCR*** for S1P and one or more other lysophospholipids, but the signals evoked by S1P alone are far weaker than those transduced by Edg-3 and Edg-5. Similarities of the structures of genes for the respective homology clusters supports this tentative classification of the Edg protein GPCRs. Future research will be directed to completion of the elucidation of genomic organization and signaling pathways, and a greater understanding of the breadth of functional roles of Edg proteins in development and activities of the nervous, cardiovascular, endocrine and immune systems.

L5 ANSWER 2 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 2000329298 EMBASE <<LOGINID::20060727>>

TI The Kaposi's sarcoma-associated herpes virus G protein-coupled receptor up-regulates vascular ***endothelial*** growth factor expression and secretion through mitogen-activated protein kinase and p38 pathways acting on hypoxia-inducible factor 1.alpha..

AU Sodhi A.; Montaner S.; Patel V.; Zohar M.; Bais C.; Mesri E.A.; Gutkind J.S.

CS J.S. Gutkind, Oral and Pharyngeal Cancer Branch, Natl. Inst. of Dent./Craniofac. Res., NIH, 30 Convent Drive, Bethesda, MD 20892-4330, United States. SG39v@nih.gov

SO Cancer Research, (1 Sep 2000) Vol. 60, No. 17, pp. 4873-4880.

Refs: 53

ISSN: 0008-5472 CODEN: CNREAB

CY United States

DT Journal; Article

FS 016 Cancer

LA English

SL English

ED Entered STN: 5 Oct 2000

Last Updated on STN: 5 Oct 2000

AB The elucidation of the molecular mechanisms governing the transition from a nonangiogenic to an angiogenic phenotype is central for understanding and controlling malignancies. Viral oncogenes represent powerful tools for disclosing transforming mechanisms, and they may also afford the possibility of investigating the relationship between transforming pathways and angiogenesis. In this regard, we have recently observed that a constitutively active G protein-coupled receptor (***GPCR***) encoded by the Kaposi's sarcoma-associated herpes virus (KSHV)/human herpes virus 8 is oncogenic and stimulates angiogenesis by increasing the secretion of vascular ***endothelial*** growth factor (VEGF), which is a key angiogenic stimulator and a critical mitogen for the development of Kaposi's sarcoma. Here we show that the KSHV ***GPCR*** enhances the expression of VEGF by stimulating the activity of the transcription factor hypoxia-inducible factor (HIF)-1.alpha., which activates transcription from a hypoxia response element within the 5'-flanking region of the VEGF promoter. Stimulation of HIF-1.alpha. by the KSHV ***GPCR*** involves the phosphorylation of its regulatory/inhibitory domain by the p38 and mitogen-activated protein kinase (MAPK) signaling pathways, thereby enhancing its transcriptional activity. Moreover, specific inhibitors of the p38 (SKF86002) and MAPK (PD98059) pathways are able to inhibit the activation of the transactivating activity of HIF-1.alpha. induced by the KSHV ***GPCR***, as well as the VEGF expression and secretion in cells overexpressing this receptor. These findings suggest that the KSHV ***GPCR*** oncogene subverts convergent physiological pathways leading to angiogenesis and provide the first insight into a mechanism whereby growth factors and oncogenes acting upstream from MAPK, as well as inflammatory cytokines and cellular stresses that activate p38, can interact with the hypoxia-dependent machinery of angiogenesis. These results may also help to identify novel targets for the development of antiangiogenic therapies aimed at the treatment of Kaposi's sarcoma and other neoplastic diseases.

L5 ANSWER 3 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 2000167620 EMBASE <<LOGINID::20060727>>

TI Sphingosine-1-phosphate signaling via the EDG-1 family of G-protein-coupled receptors.

AU Hla T.; Lee M.-J.; Ancellin N.; Thangada S.; Liu C.H.; Kluk M.; Chae S.-S.; Wu M.-T.

CS T. Hla, Center for Vascular Biology, Department of Physiology, University Connecticut Health Ctr., Farmington, CT 06030-3501, United States. hla@sun.uchc.edu

SO Annals of the New York Academy of Sciences, (2000) Vol. 905, pp. 16-24.

Refs: 50

ISSN: 0077-8923 CODEN: ANYAA

CY United States

DT Journal; Conference Article

FS 018 Cardiovascular Diseases and Cardiovascular Surgery

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 25 May 2000

Last Updated on STN: 25 May 2000

AB The bioactive lipid sphingosine-1-phosphate (SPP) is abundantly formed and released during the activation of platelets by thrombotic stimuli. Once exported, SPP interacts with the G-protein-coupled receptors (***GPCR***) of the EDG-1 family. SPP binds to EDG-1 with the dissociation constant of .apprx.8 nM and induces signal transduction events such as mitogen-activated protein kinase (MAP kinase) activation, decrease of cAMP

levels, remodeling of the actin cytoskeleton, among others. EDG-1 is a prototypical member of a large family of GPCRs that interact with glycerol and sphingolipid phosphates, namely, SPP and lysophosphatidic acid (LPA). Three other GPCRs, trivially termed EDG-3, EDG-5, and EDG-8, are also high-affinity receptors for SPP. The four SPP receptor subtypes regulate different intracellular signal transduction pathways. In vascular ***endothelial*** cells, cooperative signaling between EDG-1 and EDG-3 subtypes of SPP receptors results in adherens junction assembly, cell survival, morphogenesis into capillary-like networks, and angiogenesis. SPP acts distinctly, albeit cooperatively, with polypeptide angiogenic factors, resulting in the formation of mature neovessels. Thus SPP signaling as an extracellular mediator via the EDG-1 family of GPCRs may be a heretofore unrecognized mechanism for the regulation of angiogenesis and vascular ***endothelial*** cell function.

L5 ANSWER 4 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 1999389027 EMBASE <<LOGINID::20060727>>

TI Kaposi's sarcoma-associated herpesvirus-encoded G protein-coupled receptor activation of c-Jun amino-terminal kinase/stress-activated protein kinase and Lyn kinase is mediated by related adhesion focal tyrosine kinase/proline-rich tyrosine kinase 2.

AU Munshi N.; Ganju R.K.; Avraham S.; Mesri E.A.; Groopman J.E.

CS J.E. Groopman, Division of Experimental Medicine, Harvard Institutes of Medicine, Beth Israel Deaconess Medical Center, 4 Blackfan Circle, Boston, MA 02115, United States

SO Journal of Biological Chemistry, (5 Nov 1999) Vol. 274, No. 45, pp. 31863-31867.

Refs: 48

ISSN: 0021-9258 CODEN: JBCHA3

CY United States

DT Journal; Article

FS 016 Cancer

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 2 Dec 1999

Last Updated on STN: 2 Dec 1999

AB The Kaposi's sarcoma-associated herpesvirus (KSHV) (also known as human herpesvirus 8) has been implicated in the pathogenesis of Kaposi's sarcoma and B cell primary effusion lymphomas. KSHV encodes a G protein-coupled receptor (***GPCR***) that acts as an oncogene and constitutively activates two protein kinases, c-Jun amino-terminal kinase (JNK)/stress-activated protein kinase (SAPK) and p38 mitogen-activated protein kinase. It also induces the production of vascular ***endothelial*** growth factor. These processes are believed to be important in KSHV- ***GPCR*** -related oncogenesis. We have characterized the signaling pathways mediated by KSHV- ***GPCR*** in a reconstituted 293T cell model in which the related adhesion focal tyrosine kinase (RAFTK) was ectopically expressed. RAFTK has been shown to play an important role in growth factor signaling in endothelium and in B cell antigen receptor signaling in B lymphocytes. KSHV- ***GPCR*** induced the tyrosine phosphorylation of RAFTK. Expression of wild-type RAFTK enhanced ***GPCR*** -mediated JNK/SAPK activation, whereas dominant-negative mutant constructs of RAFTK, such as K457A (which lacks kinase activity) and Y402F (a Src-binding mutant), inhibited KSHV- ***GPCR*** -mediated activation of JNK/SAPK. RAFTK also mediated the KSHV- ***GPCR*** -induced activation of Lyn, a Src family kinase. However, RAFTK did not mediate the activation of p38 mitogen-activated protein kinase induced by KSHV. ***GPCR*** . Human interferon .gamma.-inducible protein-10, which is known to inhibit KSHV- ***GPCR*** activity, was found to reduce RAFTK phosphorylation and JNK/SAPK activation. These results suggest that in cells expressing RAFTK/proline-rich tyrosine kinase 2, such as ***endothelial*** and B cells, RAFTK can act to enhance KSHV- ***GPCR*** -mediated downstream signaling to transcriptional regulators such as JNK/SAPK.

L5 ANSWER 5 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 1999182471 EMBASE <<LOGINID::20060727>>

TI Lysophospholipid enhancement of human T cell sensitivity to diphtheria toxin by increased expression of heparin-binding epidermal growth factor.

AU Goetzl E.J.; Kong Y.; Kenney J.S.

CS Dr. E.J. Goetzl, Immunology and Allergy, Univ. of California Medical Center, Box 0711, 533 Parnassus, San Francisco, CA 94143-0711, United States

SO Proceedings of the Association of American Physicians, (1999) Vol. 111, No. 3, pp. 259-269.

Refs: 28

ISSN: 1081-650X CODEN: PAAPFD

CY United States

DT Journal; Article

FS 026 Immunology, Serology and Transplantation

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 10 Jun 1999

Last Updated on STN: 10 Jun 1999

AB The effects of lysophosphatidic acid (LPA) and sphingosine 1-phosphate (S1P) on T cell expression of heparin-binding epidermal growth factor-like growth factor (HB-EGF), the diphtheria toxin (DT) receptor, were investigated in the Tsup-1 cultured line of human CD4+ 8+ 3(low) T lymphoblastoma cells. Tsup-1 cells bear ***endothelial***

differentiation gene (edg)-2 and -4-encoded G protein-coupled receptors (GPCRs) for LPA and Edg-3 and -5 GPCRs for S1P. Suppression by DT of Tsup-1 cell protein synthesis was enhanced by LPA and S1P, with lipid structural specificity similar to that required for their recognition by Edg receptors. LPA and S1P increased the Tsup-1 cell level of immunoreactive HB-EGF, and neutralizing antibodies to HB-EGF inhibited LPA and S1P enhancement of Tsup-1 cell susceptibility to DT. Stabilized transfection of Tsup-1 cells with a combination of plasmids encoding Edg-2 plus -4 antisense mRNA suppressed the levels of Edg-2 and -4, but not Edg-3 and -5, in Western blots and reduced in parallel the increments in HB-EGF and susceptibility to DT evoked by LPA but not S1P. Similar transfection with Edg-3 plus -5 antisense plasmids suppressed Tsup-1 cell levels of immunoreactive Edg-3 and -5, but not Edg-2 or -4, and concurrently reduced S1P-, but not LPA-, induced Tsup-1 cell increases in both HB-EGF and susceptibility to DT. Edg ***GPCR***-mediated LPA and S1P enhancement of T cell sensitivity to DT, thus, may be attributable to increased expression of the DT receptor HB-EGF.

L5 ANSWER 6 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 1998196919 EMBASE <<LOGINID::20060727>>

TI Cloning of cDNAs encoding G protein-coupled receptor expressed in human ***endothelial*** cells exposed to fluid shear stress.

AU Takada Y.; Kato C.; Kondo S.; Korenaga R.; Ando J.
CS Y. Takada, Institute for Life Science Research, Asahi Chemical Industry Co., Ltd., 2-1 Samejima, Fuji-City, Shizuoka 416, Japan.

a8886784@ut.asahi-kasei.co.jp
SO Biochemical and Biophysical Research Communications, (26 Nov 1997) Vol. 240, No. 3, pp. 737-741.

Refs: 20

ISSN: 0006-291X CODEN: BBRCA

CY United States

DT Journal; Article

FS 022 Human Genetics

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 2 Jul 1998

Last Updated on STN: 2 Jul 1998

AB A cDNA library of human umbilical vein ***endothelial*** cells exposed to fluid shear stress was constructed to search for functional ***endothelial*** genes expressed under flow conditions, and cDNAs encoding members of the G protein-coupled receptor (***GPCR***) family were cloned by a polymerase chain reaction (PCR) method using degenerate oligonucleotide primers. One of the two ***GPCR*** clones obtained was edg-1, and the other clone is a novel gene named FEG-1 that encodes a 375-amino acid protein similar to the receptors for both angiotensin II and chemokines. Reverse transcriptase PCR showed that the FEG-1 and edg-1 mRNA levels in ***endothelial*** cells increased markedly in response to fluid flow. This suggests that FEG-1 and edg-1 may be receptor genes that play important roles in the regulation of ***endothelial*** function under physiological blood flow conditions.

L5 ANSWER 7 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 96137136 EMBASE <<LOGINID::20060727>>

DN 1996137136

TI .alpha.1-Adrenergic receptor subtypes: Molecular structure, function, and signaling.

AU Graham R.M.; Perez D.M.; Hwa J.; Piascik M.T.

CS Victor Chang Cardiac Res. Institute, 376 Victoria St, Darlinghurst, NSW 2010, Australia

SO Circulation Research, (1996) Vol. 78, No. 5, pp. 737-749.

ISSN: 0009-7330 CODEN: CIRUAL

CY United States

DT Journal; (Short Survey)

FS 030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 20 May 1996

Last Updated on STN: 20 May 1996

AB Recent insights into .alpha.1AR biology have confirmed the heterogeneity of this important class of signaling molecules and have identified enormous diversity in the signaling pathways used by .alpha.1ARs in regulating cellular functions. Although initially confounding our understanding of .alpha.1ARs, the molecular cloning of the various .alpha.1 subtypes has clearly contributed greatly to these insights. Much remains to be learned, however, about the molecular mechanisms of receptor activation, the regulation of receptor expression, and the involvement of .alpha.1ARs in physiology and disease. It is also of interest to speculate why there are multiple .alpha.1ARs or, more generally, multiple subtypes of many members of the ***GPCR*** superfamily. For AR subtypes, this issue has been considered recently in an interesting commentary by Milligan et al. This article, however, focused mainly on the possible reasons for having both .alpha.- and .beta.-adrenergic receptors rather than on the implications of multiple .alpha.1AR subtypes. With respect to the former, Milligan et al correctly point out that adrenergic control of cardiac function can no longer be considered to be exclusively by .beta.1ARs, since .alpha.1ARs, .alpha.2ARs, .beta.2ARs, and .beta.3ARs have been identified on cardiac myocytes. .alpha.1AR responses appear not to be as important in the normal heart as in disease states, where the heart is altered in favor of .alpha.1ARs and .beta.2ARs. This

switch in adrenergic responsiveness may provide a backup for maintaining cardiac function in the event of .beta.1AR failure. Thus, increased .alpha.1 responsiveness is observed during chronic treatment with .beta.1 antagonists in cardiac ischemia, cardiac hypertrophy, hypothyroidism, and diabetes. In blood vessels, .alpha.1AR and .beta.1AR allow sympathetic control of both vascular smooth muscle contraction and dilation, respectively. .alpha.1AR subtypes are present throughout the vasculature but are more prominent on the arterial side, where they regulate peripheral resistance. Venous tone, prejunctional modulation of sympathetic activity, and ***endothelial*** release of endothelium-derived relaxing factor, on the other hand, are mediated predominantly by .alpha.2ARs. With respect to the multiplicity of .alpha.1AR subtypes, it is of interest that all three subtypes recognize the endogenous catecholamines norepinephrine and epinephrine with similar affinity, even though they can be distinguished by their differential binding of a variety of synthetic agonists and antagonists. This finding and the fact that all subtypes appear to activate all effector pathways similarly (note that thus far only activation of PLC, PLA2, and PLD have been examined in detail for all subtypes) suggest that from the point of view of the organism, the various subtypes would be indistinguishable. Our ability to discriminate these subtypes may thus merely be due to the development of synthetic ligands that can detect subtle differences between their ligand recognition sites that are functionally inconsequential. If this hypothesis is correct, then it implies that teleologically the major functional consequences of the various subtypes lie not in their activation or signaling but in their ability to be differentially expressed during development (in response to various physiological or pathophysiological stimuli) and/or in various tissues and species. As a corollary, one can predict that the elements involved in the regulation of receptor gene expression will differ between the subtypes. From a pharmacological point of view, however, the potential for developing compounds that are highly selective for an individual subtype remains of considerable therapeutic significance.

L5 ANSWER 8 OF 13 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 96050064 EMBASE <<LOGINID::20060727>>

DN 1996050064

TI cDNA cloning of a novel G-protein-coupled receptor with a large extracellular loop structure.

AU Roglic A.; Prossnitz E.R.; Cavanagh S.L.; Pan Z.; Zou A.; Ye R.D.

CS Department of Immunology, The Scripps Research Institute, La Jolla, CA 92037, United States

SO Biochimica et Biophysica Acta - Gene Structure and Expression, (1996) Vol. 1305, No. 1-2, pp. 39-43.

ISSN: 0167-4781 CODEN: BBGSD5

CY Netherlands

DT Journal; Article

FS 021 Developmental Biology and Teratology

022 Human Genetics

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 5 Mar 1996

Last Updated on STN: 5 Mar 1996

AB A cDNA designated as AZ3B has been isolated from a differentiated HL-60 cell cDNA library with a probe derived from the N-formyl peptide receptor gene. The 1.97-kb cDNA encodes a novel G protein-coupled receptor (***GPCR***) with 482 amino acids. In addition to the predicted 7 transmembrane domains common to all GPCRs, the protein encoded by AZ3B contains a large extracellular loop of approx. 172 amino acids between the fourth and the fifth transmembrane domains, a feature unique among the hundreds of GPCRs identified to date. High sequence homology exists between the AZ3B protein and a number of chemoattractant receptors in the amino-terminal 170 residues and the carboxyl-terminal 150 residues. Northern and flow cytometric analyses suggested that the AZ3B message and protein are widely expressed in several differentiated hematopoietic cell lines, in the lung, placenta, heart, and ***endothelial*** cells. We postulate that the AZ3B protein defines a distinct group of receptors within the ***GPCR*** superfamily.

L5 ANSWER 9 OF 13 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 2001:68845 BIOSIS <<LOGINID::20060727>>

DN PREV200100068845

TI Expression of EDG receptors on cultured rat cardiomyocytes and their role in mediating sphingosine-1-phosphate calcium deregulation.

AU Sabbadini, Roger A. [Reprint author]; Nakajima, Nobuko [Reprint author];

Cavalli, Amy L. [Reprint author]; Ligutti, Joseph A. [Reprint author];

Giembsolski, Christopher C. [Reprint author]; McDonough, Patrick M.

[Reprint author]; Betto, Romeo; Sandona, Dorian; Palade, Philip T.;

Dettbarn, Christine A.; Klepper, Robert E.

CS San Diego State Univ, San Diego, CA, USA

SO Circulation, (***October 31, 2000***) Vol. 102, No. 18 Supplement, pp.

II, 275. print.

Meeting Info.: Abstracts from American Heart Association Scientific

Sessions 2000. New Orleans, Louisiana, USA, November 12-15, 2000.

American

Heart Association.

CODEN: CIRCAZ. ISSN: 0009-7322.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 31 Jan 2001
Last Updated on STN: 12 Feb 2002

L5 ANSWER 10 OF 13 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 2000:386680 BIOSIS <<LOGINID::20060727>>

DN PREV200000386680

TI Identification of an EDG7 variant, HOFNH30, a G-protein-coupled receptor for lysophosphatidic acid.

AU Fitzgerald, Laura Rydelek; Dytko, George M.; Sarau, Henry M.; Mannan, Ishrat Jahan; Ellis, Catherine; Lane, Pamela A.; Tan, Kong B.; Murdock, Paul R.; Wilson, Shelagh; Bergsma, Derk J.; Ames, Robert S.; Foley, James J.; Campbell, David A.; McMillan, Lynnette; Evans, Nicholas; Elshourbagy, Nabil A.; Minehart, Heather; Tsui, Ping [Reprint author]

CS Department of Molecular Biology, SmithKline Beecham Pharmaceuticals, 709 Swedeland Road, Mail code UE0548, King of Prussia, PA, 19406, USA

SO Biochemical and Biophysical Research Communications, (***July 14, ***
*** 2000***) Vol. 273, No. 3, pp. 805-810. print.
CODEN: BBRC9A. ISSN: 0006-291X.

DT Article

LA English

OS Genbank-AF127138; Genbank-AF236117; Genbank-NM004720; Genbank-U80811

ED Entered STN: 13 Sep 2000

Last Updated on STN: 8 Jan 2002

AB We have identified a cDNA, designated HOFNH30, which encodes a 354 amino

acid G-protein-coupled receptor (***GPCR***). This receptor has 96% amino acid identity to the Jurkat-T cell-derived EDG7 and could be a splice variant. RT-PCR analysis demonstrated that HOFNH30 mRNA is expressed in placenta whereas EDG7 mRNA shows highest expression in prostate. The HOFNH30 gene is localized to human chromosome 1p22.3-1p31.1. When HOFNH30 was expressed in RBL-2H3 cells, LPA and phosphatidic acid (PA) induced a calcium mobilization response with EC50 values of 13 nM and 3 muM, respectively. LPA also induced phosphorylation of mitogen-activated protein kinase (p42MAPK and p44MAPK) in HOFNH30-transfected but not vector-transfected RBL-2H3 cells. In the present study, we have identified a novel variant from the EDG receptor family, a ***GPCR*** for which LPA is a high-affinity endogenous ligand.

L5 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2000:806240 CAPLUS <<LOGINID::20060727>>

DN 134:98321

TI A subfamily of G protein-coupled cellular receptors for lysophospholipids and lysosphingolipids

AU Goetzl, Edward J.; An, Songzhu

CS Departments of Medicine and Microbiology-Immunology, University of California Medical Center, San Francisco, CA, 94143-0711, USA

SO Advances in Experimental Medicine and Biology (***1999***), 469/Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation, and Radiation Injury, 4), 259-264
CODEN: AEMBAP; ISSN: 0065-2598

PB Kluwer Academic/Plenum Publishers

DT Journal; General Review

LA English

AB A review with 12 refs. The results of mol. cloning and homol. searches have identified a min. of five different proteins of the ***endothelial*** differentiation gene (edg) encoded subfamily of GPCRs. Edg protein GPCRs show amino acid sequence identity of 31% to 34% as a subfamily, but contain two homol. clusters with greater similarity of structures and functions. One cluster of high amino acid sequence homol. includes Edg-2 and Edg-4 proteins, that encode GPCRs for LPA, but not lysosphingolipids. A second homol. cluster encompasses Edg-1, Edg-3 and Edg-5. Edg-3 and Edg-5 encode GPCRs specific for sphingosine 1-phosphate (S1P), but not lysophosphatidic acid (LPA). Preliminary data suggest that, Edg-1 encodes a ***GPCR*** for S1P and one or more other lysosphingolipids, but the signals evoked by S1P alone are far weaker than those transduced by Edg-3 and Edg-5. Similarities of the structures of genes for the resp. homol. clusters supports this tentative classification of the Edg protein GPCRs. Future research will be directed to completion of the elucidation of genomic organization and signaling pathways, and a greater understanding of the breadth of functional roles of Edg proteins in development and activities of the nervous, cardiovascular, endocrine and immune systems.

RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1999:456506 CAPLUS <<LOGINID::20060727>>

DN 131:224228

TI Identification of a spliced gene from Kaposi's sarcoma-associated herpesvirus encoding a protein with similarities to latent membrane proteins 1 and 2A of Epstein-Barr virus

AU Glenn, Mark; Rainbow, Lucille; Aurade, Frederic; Davison, Andrew; Schulz, Thomas F.

CS Molecular Virology Group, Department of Medical Microbiology and Genito-Urinary Medicine, University of Liverpool, Liverpool, L69 3GA, UK

SO Journal of Virology (***1999***), 73(8), 6953-6963

CODEN: JOVIAM; ISSN: 0022-538X

PB American Society for Microbiology

DT Journal

LA English

AB Kaposi's sarcoma-assocd. herpesvirus (KSHV) or human herpesvirus 8 (HHV-8)

is a novel herpesvirus implicated as the causative agent of Kaposi's sarcoma (KS), primary effusion lymphoma, and some cases of multicentric Castleman's disease. KSHV persists in the majority of KS spindle (***endothelial*** tumor) cells and lymphoid cells in a latent form, with only a limited set of viral genes expressed in a tissue-specific manner. Here, we report the identification of a family of alternatively-spliced transcripts of approx. 7.5 kb expressed in latently infected body cavity-based lymphoma (BCBL) cell lines which are predicted to encode membrane proteins with similarities to the LMP2A and LMP1 proteins of Epstein-Barr virus. In two highly divergent sequence variants of the right end of the KSHV genome, alternative splicing of eight exons located between KSHV ORF 75 and the terminal repeats yields transcripts appropriate for proteins with up to 12 transmembrane domains, followed by a hydrophilic C-terminal, presumably cytoplasmic, domain. This C-terminal domain contains several YxxL/L motifs reminiscent of LMP2A and a putative TRAF binding site as in LMP1. In latently (persistently) infected BCBL cells the predominant transcript utilizes all eight exons, whereas in phorbol-ester-induced cells, a shorter transcript, lacking exons 4 and 5, is also abundant. We also found evidence for an alternative use of exon 1. Transfection of an epitope-tagged cDNA construct contg. all exons indicates that the encoded protein is localized on cell surface and intracellular membranes, and glutathione S-transferase pull-down expts. indicate that its cytoplasmic domain, like that of LMP1, interacts with TRAF1, -2, and -3. Two of 20 KS patients had antibodies to the hydrophilic C-terminal domain, suggesting that the protein is expressed in vivo.

RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1998:745196 CAPLUS <<LOGINID::20060727>>

DN 130:11308

TI Cloning and cDNA sequences of two human G protein-coupled receptors: EBV-induced ***GPCR*** 2 (EBI-2) and EDG-1-like ***GPCR***

IN Ruben, Steven M.; Li, Yi

PA Human Genome Sciences, Inc., USA

SO PCT Int. Appl., 65 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850549	A2	19981112	WO 1998-US9048	19980507 <--
WO 9850549	A3	20000406		
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6060272	A	20000509	US 1997-852824	19970507 <--
CA 2289046	AA	19981112	CA 1998-2289046	19980507 <--
EP 1007670	A2	20000614	EP 1998-920965	19980507 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002508657	T2	20020319	JP 1998-548332	19980507
EP 1369430	A2	20031210	EP 2003-15456	19980507
EP 1369430	A3	20040128		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
US 6887683	B1	20050503	US 2000-518383	20000303
CA 2307709	AA	20011105	CA 2000-2307709	20000505
US 2002052043	A1	20020502	US 2001-827937	20010409
US 2005123961	A1	20050609	US 2004-968990	20041021
PRAI US 1997-852824	A	19970507		
EP 1998-920965	A3	19980507		
WO 1998-US9048	W	19980507		
US 2000-518381	B1	20000303		
US 2000-518383	A1	20000303		

AB Two human G-protein coupled receptor polypeptides and DNA (RNA) encoding

each of such polypeptides and a procedure for producing such polypeptides by recombinant techniques is disclosed. The cDNA for Epstein-Barr virus-induced G protein-coupled receptor (EBI-2) comprises 2249 bp encoding a protein 342 amino acids in length with 25% identity and 49% similarity to the amino acid sequence of human EBI-1, whereas the cDNA for EDG-1-like receptor comprises 1637 bp encoding a protein 260 amino acids in length with 54% identity and 73% similarity to the amino acid sequence of human EDG-1 orphan G protein-coupled receptor. EBI-2 mRNA was discovered in a cDNA library derived from umbilical vein ***endothelial*** cells, and may also be found in neutrophil leukocyte cells and corpus colosum cells. EDG-1-like receptor mRNA was initially found an activated neutrophil cDNA library, cyclohexamine-treated Raji cells, the RSR;11 bone marrow cell line, activated T-cells, tonsils, and CD34-pos. cord blood cells. Also disclosed are methods for utilizing such polypeptides for identifying antagonists and agonists to such polypeptides. Also disclosed are diagnostic methods for detecting a mutation in the nucleic acid sequence of each of the G-protein coupled receptors.

=> s Galvin, K7/au
L6 283 GALVIN, K7/AU

=> s Rudolph Owen, L7/au
L7 76 RUDOLPH OWEN, L7/AU

=> s l6 and GPCR
L8 4 L6 AND GPCR

=> s l7 and GPCR
L9 2 L7 AND GPCR

=> s l8 or l9
L10 5 L8 OR L9

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11 5 DUP REM L10 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 5 ANSWERS - CONTINUE? Y/(N):y

L11 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:320028 CAPLUS <<LOGINID::20060727>>
DN 138:332935
TI Protein and cDNA sequences of a human G protein-coupled protein receptor sequence homolog and therapeutic use
IN Logan, Thomas Joseph; ***Galvin, Katherine M.***
PA Millennium Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 134 pp.
CODEN: PIXXD2

DT Patent
LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003033661	A2	20030424	WO 2002-US32974	20021016
WO 2003033661	A3	20031002		
WO 2003033661	C1	20031224		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003109044	A1	20030612	US 2002-267811	20021009
EP 1435975	A2	20040714	EP 2002-780469	20021016
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-329648P	P	20011016		
WO 2002-US32974	W	20021016		

AB The invention provides protein and cDNA sequences of a human protein, designated 279, which has sequence homol. with human G protein-coupled receptor (***GPCR***) family members. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. 279 nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 279 gene has been introduced or disrupted. The invention still further provides isolated 279 proteins, fusion proteins, antigenic peptides and anti-279 antibodies. Methods utilizing compns. of the invention to treat, prevent or diagnose angiogenic disorders, e.g., cardiovascular and cancerous disorders, are also provided.

L11 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:912729 CAPLUS <<LOGINID::20060727>>
DN 139:376249
TI Novel 10 human G-protein coupled receptor genes 18636, 2466, 43238, 1983, 52881, 2398, 45449, 50289, 52872 and 26908, and therapeutic uses therefor
IN Glucksmann, Maria A.; Silos-Santiago, Inmaculada; Carroll, Joseph M.; ***Galvin, Katherine M.***

PA Millennium Pharmaceuticals, Inc., USA
SO U.S. Pat. Appl. Publ., 163 pp., Cont.-in-part of U.S. Ser. No. 282,837.
CODEN: USXXCO

DT Patent
LA English

FAN.CNT 16

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003215860	A1	20031120	US 2003-407079	20030403
US 2002061522	A1	20020523	US 2001-796338	20010228
US 2002039762	A1	20020404	US 2001-863200	20010522
US 2003064399	A1	20030403	US 2002-225094	20020821
US 2003087281	A1	20030508	US 2002-226102	20020822
US 2003124670	A1	20030703	US 2002-272417	20021015
US 2003082738	A1	20030501	US 2002-282837	20021029
PRAI US 2000-186059P	P	20000229		
US 2000-191845P	P	20000324		
US 2000-206019P	P	20000522		

US 2000-715790	B1	20001117
US 2001-796338	B1	20010228
US 2001-863200	B2	20010522
US 2001-314041P	P	20010822
US 2001-314185P	P	20010822
US 2002-225094	A2	20020821
US 2002-226102	A2	20020822
US 2002-272417	A2	20021015
US 2002-282837	A2	20021029

AB The invention provides protein and cDNA sequences of 10 isolated G-protein coupled receptor genes, designated 18636, 2466, 43238, 1983, 52881, 2398, 45449, 50289, 52872 and 26908. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. above ***GPCR*** nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an above ***GPCR*** gene has been introduced or disrupted. The invention still further provides isolated ***GPCR*** proteins, fusion proteins, antigenic peptides and specific antibodies. Diagnostic and therapeutic methods utilizing compns. of the invention are also provided.

L11 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:58706 CAPLUS <<LOGINID::20060727>>
DN 138:132217

TI Human nucleic acid sequences encoding G-protein coupled receptors and their recombinant production and use in diagnostic methods
IN Glucksmann, Maria Alexandra; Hodge, Martin R.; Hunter, John J.; ***Rudolph-Owen, Laura A.***; Silos-Santiago, Inmaculada; Weich, Nadine S.

PA Millennium Pharmaceuticals, Inc., USA
SO U.S. Pat. Appl. Publ., 149 pp., Cont.-in-part of U.S. Ser. No. 741,783.
CODEN: USXXCO

DT Patent

LA English

FAN.CNT 6

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003017539	A1	20030123	US 2002-165844	20020607
US 2003162172	A1	20030828	US 2000-741783	20001218
PRAI US 1998-88857	B2	19980602		
US 1998-145745	A2	19980902		
US 1999-234923	A2	19990121		
US 1999-324465	A2	19990602		
US 1999-340880	A2	19990628		
US 1999-383745	A2	19990826		
US 1999-464685	A2	19991216		
US 2000-741783	A2	20001218		

AB The invention provides 4 isolated cDNA mols. that encode novel human polypeptides with sequence similarity to G protein-coupled receptors. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. the nucleic acid mols. of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a sequence of the invention has been introduced or disrupted. The invention still further provides isolated proteins, fusion proteins, antigenic peptides, and antibodies. Diagnostic methods utilizing compns. of the invention are also provided.

L11 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:798478 CAPLUS <<LOGINID::20060727>>
DN 135:340279

TI A novel G protein-coupled receptor sequence homolog 4941, and related methods and compositions for the diagnosis and treatment of cardiovascular and tumorigenic disease

IN ***Galvin, Katherine A.***; ***Rudolph-Owen, Laura A.***

PA Millennium Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 118 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001081634	A2	20011101	WO 2001-US13788	20010425
WO 2001081634	A3	20020912		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001057406	A5	20011107	AU 2001-57406	20010425
EP 1280937	A2	20030205	EP 2001-930917	20010425
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004091929	A1	20040513	US 2003-696706	20031029
PRAI US 2000-199908P	P	20000426		
US 2000-635521	A	20000809		
WO 2001-US13788	W	20010425		

AB The invention provides protein and cDNA sequences for a novel human G protein-coupled receptor (***GPCR***) sequence homolog 4941.

GPCR 4941 gene is located at human chromosome 2q21-22 and its mRNA

tissue profile is provided. Specifically, the present invention identifies ***GPCR*** 4941 genes which are differentially expressed in cardiovascular disease states, relative to their expression in normal, or non-cardiovascular disease states, and/or in response to manipulations relevant to cardiovascular disease. The present invention also identifies ***GPCR*** 4941 genes as differentially expressed in tumorigenic disease, e.g., ovarian cancer. The present invention relates to methods and compns. for the diagnosis and treatment of cardiovascular disease and cancers. These diseases include but not limit, atherosclerosis reperfusion injury, hypertension, restenosis, arterial inflammation, and endothelial cell disorders, such as disorders assocd. with aberrant endothelial cell growth, angiogenesis and/or vascularization, e.g., tumorigenic disorders. The present invention describes methods for the diagnostic evaluation and prognosis of various cardiovascular and tumorigenic diseases, and for the identification of subjects exhibiting a predisposition to such conditions. The present invention provides methods for the diagnostic monitoring of patients undergoing clin. evaluation for the treatment of cardiovascular disease and tumorigenic, and for monitoring the efficacy of compds. in clin. trials. The present invention also provides methods for the identification and therapeutic use of compds. as treatments of cardiovascular and tumorigenic disease.

L11 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2001:598174 CAPLUS <<LOGINID::20060727>>

DN 135:176475

TI Polynucleotides encoding protein 39406, a seven transmembrane protein, and their diagnostic and therapeutic uses

IN Glucksmann, Maria Alexandra; ***Galvin, Katherine M.***

PA Millennium Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 125 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001059109	A1	20010816	WO 2001-US4074	20010208
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, FR, GB, GD, GE, GH, GM, GR, GU, HA, HE, HO, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, ME, MG, MK, MN, MW, MX, MY, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2001044130	A1	20011122	US 2001-779239	20010208
PRAI US 2000-180912P	P	20000208		

AB The present invention relates to a newly identified seven-transmembrane protein, potentially a receptor belonging to the superfamily of G-protein-coupled receptors. The invention also relates to polynucleotides encoding the protein, named protein 39406. The invention further relates to methods using the polypeptides and polynucleotides as a target for diagnosis and treatment in protein 39406-mediated or -related disorders. The invention further relates to drug-screening methods using the polypeptides and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the polypeptides and polynucleotides. The invention further relates to procedures for producing the polypeptides and polynucleotides. CDNA for human protein 39406 was cloned based on an expressed sequence tag (EST) that had sequence homol. to G protein-coupled receptor (***GPCR***) sequences. Sequence anal. of the assembled cDNA revealed a novel putative seven transmembrane protein with homol. to the rhodopsin family of GPCRs and P2Y-like GPCRs. MRNAs for protein 39406 were detected in many normal and diseased human tissues by RT-PCR.

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=>

--Logging off of STN--

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		83.63	96.44
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)			
TOTAL			
CA SUBSCRIBER PRICE		-6.75	-6.75